

Complications of Stenting for Symptomatic Middle Cerebral Artery Stenosis and Their Rescue

-Case Report-

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Intravascular stents are being used with increasing frequency in interventional neuroradiology. We present two cases of arterial rupture and subarachnoid hemorrhage during middle cerebral artery stenting. Extravasation of contrast material and massive subarachnoid hemorrhage during stenting in one case resulted in mortality. In the other case, overlapping stenting and balloon tamponade on the dissected vessel, however, were performed emergently. Thereafter, thrombotic occlusion of the middle cerebral artery was managed by intraarterial abciximab administration. Serial angiography showed that normal vessel patency was reestablished within 20 min. We discuss complications of stenting for symptomatic middle cerebral artery stenosis and their rescue work.

KEY WORDS : Intravascular stent · Middle cerebral artery stenosis · Dissection · Rupture · Rescue work.

Introduction

Arterial stenting is a well-established technique for the treatment of atherosclerotic lesions of the coronary, iliac, and renal vasculatures⁷⁻⁹. Its use in the cerebral arteries has been largely confined to the extracranial portions of the vessels. Recent advances in stent technology have allowed the introduction of more flexible stents that may be tracked more easily in the intracranial vessels. These systems revolutionize the management of ischemic and hemorrhagic cerebrovascular disease ; however, several issues remain before widespread application of stent technology to the intracranial circulation can be advocated. Of these problems, arterial dissections are one of most serious complications and can cause ischemic symptoms by acute thromboembolism or delayed stenosis⁵. In the worst case, arterial rupture may develop the intracranial hemorrhage and result in serious morbidity or mortality¹⁹.

As experience is gained in the intracranial circulation with these devices, unique procedural complications and potential solutions are sure to occur. We report the cases of two patients with acute arterial rupture during the stenting for the severe stenosis of the middle cerebral artery, treated immediately with balloon tamponade with placement of an additional, overlapping stent in one patient.

Case Reports

Case 1

A 54-year-old man suffered from multiple episodes of transient left-hemiparesis and speech arrest despite treatment with antiplatelet medication (aspirin 100mg/day) for 15 months. An MR angiogram suggested the diagnosis of middle cerebral artery (MCA) stenosis, which was subsequently confirmed by a diagnostic cerebral angiogram. At presentation, the patient was neurologically intact, and a baseline MR image of the brain revealed no definite evidence of infarction. Diagnostic cerebral angiography revealed a highgrade (90%), eccentric, focal (length <5mm), atherosclerotic stenosis of the right MCA (Fig. 1A). We decided to perform a stent-assisted angioplasty for middle cerebral artery stenosis due to repeated transient ischemic attacks regardless of medication.

All procedures were performed under local anesthesia to monitor his neurological status during the operation. A bolus dose of heparin (100units/kg) was administered intravenously, to obtain an activated clotting time of approximately 300 seconds, and a 7-French, straight, Envoy guiding catheter (Cordis Endovascular, Miami Lakes, FL) was placed over an exchange wire in the internal carotid artery, and the catheter was connected to a continuous heparinized saline

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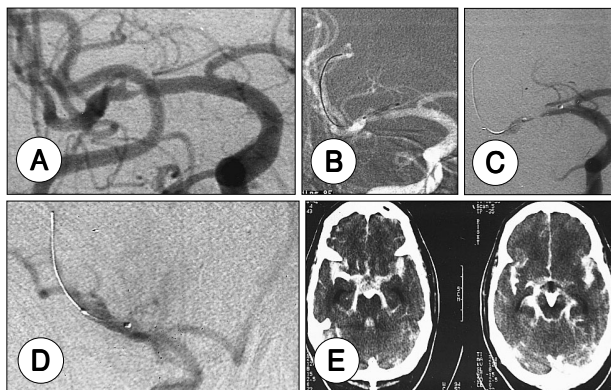


Fig. 1. Arterial rupture during stenting with GFX 3.5- \times 9.5-mm stent for the right M1 stenosis. A : Initial angiogram shows a high-grade(90%), eccentric, focal(length<5mm), atherosclerotic stenosis of the M1 segment. B : A 0.014-inch microguide wire is advanced to the stenosis of the right M1 segment, crossed the lesion, and is introduced to the level of inferior division of the right middle cerebral artery to ensure maximal support. C : Angiogram confirms its intraluminal position. D : Angiogram obtained immediately after dilatation reveals a rupture of the right M1 segment, manifested by acute extravasation of contrast material. E : CT scan demonstrates massive subarachnoid hemorrhage with diffuse distribution of contrast material.

flush. A 0.014-inch microguide wire(Wizdom ; Cordis Co., Miami, FL) was then advanced just proximal to the stenosis of the right MCA, crossed the lesion, and was introduced to the level of inferior division of the right MCA to ensure maximal support(Fig. 1B). Injection of contrast material through the catheter confirmed its intraluminal position(Fig. 1C). Thereafter, the balloon delivery apparatus, with a preloaded GFX 3.5- \times 9-mm coronary stent(Medtronic Arterial Vascular Engineering Inc., Santa Rosa, CA), was advanced over the exchange wire and positioned across the stenosis by use of roadmapping technique and deployed abruptly by inflating the balloon to 8 atm for 3 seconds. During inflation of balloon, the patient complained a sudden headache and stiff neck. Angiogram obtained immediately after dilatation revealed a rupture of the right MCA, manifested by acute extravasation of contrast material(Fig. 1D). Subsequently, the patient presented with generalized tonic-clonic seizure with mental change, controlled with 10mg diazepam(Valium) intravenously. The patient was intubated for airway maintenance and transferred to CT scan room. CT scan showed massive subarachnoid hemorrhage with diffuse distribution of contrast material(Fig. 1E). The patient was dead 3 days later.

Case 2

A 42-year-old woman suffered from multiple episodes of transient right-arm paralysis and speech arrest despite treatment with antiplatelet medication(aspirin 325mg/day) for 5

months. She had experienced headache and dizziness approximately 5 months earlier and had been found to have a borderline infarction in the left hemisphere in MR imaging and a severe stenosis of the left middle cerebral artery in MR angiograms. Three months later, she experienced right-arm weakness and verbal disturbance, but these symptoms resolved in 24 hours. Two months later, she experienced the same weakness and speech arrest and was admitted to our institution. The patient recovered completely in 10 hours and had a normal neurologic examination. Cerebral angiography confirmed the presence of 90% stenosis of the left MCA with slow flow to the distal MCA territory(Fig. 2A). Despite medical treatment, the patient experienced multiple episodes of right-sided motor weakness and speech arrest during the previous 5-month period. Therefore, the patient decided to undergo a stent-assisted angioplasty for severe stenosis of the left MCA.

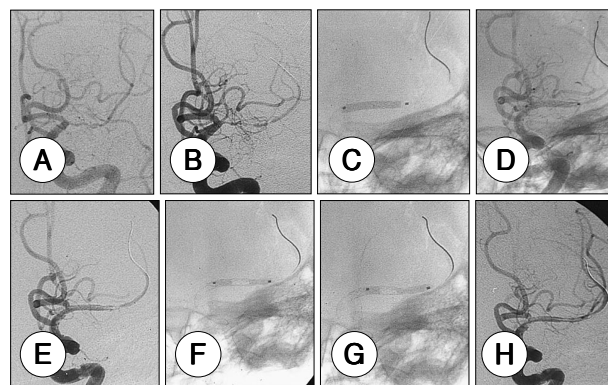


Fig. 2. Angiograms showing a severe middle cerebral artery stenosis treated with placement of S-660 2.5- \times 18-mm stent. A : Initial angiogram shows severe stenosis of the left M1 segment. B : Angiogram reveals the intraluminal position of a 0.014-inch microguide wire. C : S-660 2.5- \times 18-mm stent (Medtronic Arterial Vascular Engineering Inc., CA, USA) is deployed by inflating the balloon at 6 atm for 15 seconds. D, E : After the deployment of the stent, angiogram demonstrates remaining stenosis of the left middle cerebral artery. F : The 2.5-mm balloon is advanced to the stenotic portion of the distal middle cerebral artery and further expanded. G : The 2.5-mm balloon inflates at 2 atm for 15 seconds, and subsequently deflates. H : After balloon angioplasty, angiogram shows nearly restored arterial lumen.

The endovascular approach was performed via a transfemoral route with the patients under local anesthesia. A bolus dose of heparin(100units/kg) was administered intravenously, to obtain an activated clotting time of approximately 300 seconds, and a 7-French, straight, Envoy-guide catheter (Cordis, FL, USA) was selectively placed in the internal carotid artery, and the catheter was connected to a continuous heparinized saline flush. A Prowler 14 microcatheter (Cordis, FL, USA) was then advanced coaxially over a 0.035-inch GT wire(Terumo, Tokyo, Japan) just proximal to the stenosis of the left MCA. Then, 60,000 U of urokinase

was infused for 3 minutes through the microcatheter to reveal any evidence of acute thrombus. Posturokinase angiography revealed no change in the morphology of the lesion. The GT wire was then replaced by a 0.014-inch Hi-Torque microguide wire (Advanced Cardiovascular System, CA, USA). It was then advanced just proximal to the stenosis of the left MCA, crossed the lesion, and was introduced to the level of inferior division of the left middle cerebral artery to ensure maximal support. Injection of contrast material through the catheter confirmed its intraluminal position (Fig. 2B). Therefore, the balloon delivery apparatus, with a preloaded S-660 2.5- \times 18-mm stent (Medtronic Arterial Vascular Engineering Inc., CA, USA), was advanced over the exchange wire and positioned across the stenosis by use of roadmapping technique and deployed by inflating the balloon at 6 atm for 15 seconds (Fig. 2C). Postdeployment angiography demonstrated remaining stenosis of the left MCA (Fig. 2D, E). Therefore, the 2.5-mm balloon was advanced to the stenotic portion of the distal MCA and further expanded (Fig. 2F). The patient underwent a single balloon inflation procedure at 2 atm for 15 seconds, followed by deflation, and two repeat of the procedure for 4 minutes (Fig. 2G, H). At the fourth trial of balloon inflation with 3 atm pressure, the patient complained a sudden headache and stiff neck. Selective angiogram obtained immediately after dilatation revealed a rupture of the left MCA, manifested by acute extravasation of contrast material (Fig. 3A). The patient was treated emergently by placement of a second S-660 2.5- \times 18-mm stent (Medtronic Arterial Vascular Engineering Inc., CA, USA) within the original stent. Second stent was overlapped with the previous stent (Fig. 3B, C). Subsequently the patient underwent tamponade balloon inflation for 3 minutes, followed by rapid deflation, and a repeat of the procedure after 1 minute (Fig. 3D). The size of the balloon chosen for tamponade was 1 mm smaller in diameter than the size of the balloon that caused the rupture, and the balloon was fully inflated without the use of a manometer. No extravasation was visualized, but acute thrombotic occlusion of the distal MCA located beyond an inflated balloon was encountered (Fig. 3E). In a rescue attempt to dissolve the intraluminal thrombus, 7mg of undiluted abciximab (Reopro; Centocor, Malvern, PA) was administered through the catheter over 5 minutes. Angiography performed 10 minutes after the abciximab bolus showed partial recanalization of the left MCA thrombus (Fig. 3F). Additional 3mg of abciximab was administered over a 1 minute period. Subsequent angiography performed 20 minutes after abciximab bolus administration showed complete recanalization of the left MCA (Fig. 3G, H). There was

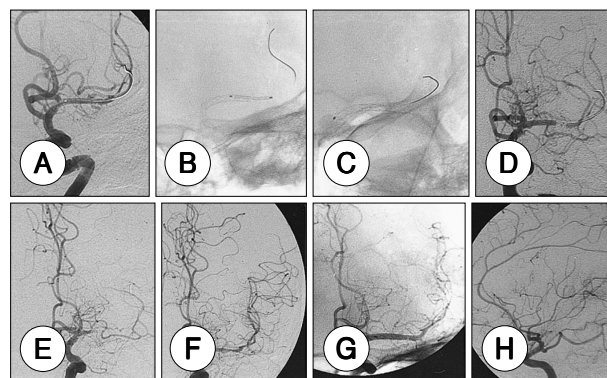


Fig. 3. Angiograms showing an arterial rupture treated with placement of an additional S-660 2.5- \times 18-mm stent and balloon tamponade, and subsequent thrombotic occlusion treated by the intraarterial administration of abciximab. A : Angiogram reveals an acute extravasation of contrast material. B, C : Fluoroscopic image shows second stent is overlapped with previous stent. D : Angiogram shows balloon tamponade on the ruptured points. E : Angiogram shows no extravasation of contrast material, but acute thrombotic occlusion of the distal M1 segment located beyond an inflated balloon. F : After the intraarterial administration of 7mg abciximab, angiogram shows a partial recanalization of the left M1 segment. G, H : After the additional administration of 3mg abciximab, angiograms reveal complete recanalization of the left middle cerebral artery.

no evidence of distal embolism on the intracranial angiograms, and the patient had no new neurologic deficits after the procedure. CT scans of the head obtained immediately after the procedure depicted subarachnoid hemorrhage on the sylvian fissure.

Full systemic heparinization was continued for 48 hours. Subsequently oral clopidogrel (75mg daily) and aspirin (300 mg daily) were begun and continued for 6 weeks. Angiography performed the next day showed MCA to be normal in appearance. The patient underwent lumboperitoneal shunt 18 days after the procedure due to communicating hydrocephalus and was discharged home 12 days after the shunt. The patient remained neurologically stable at the 7 months clinical follow-up.

Discussion

The concept of using intracranial stent-assisted angioplasty to treat medically refractory, symptomatic, intracranial, vascular stenosis was based on experience with cardiac intervention. The use of stents for the treatment of intracranial disease has previously been limited by rigid stent and stent delivery catheter designs that are unable to atraumatically maneuver acute vascular bends. Stent technology is advancing at a rapid pace, with many flexible stent delivery systems now capable of reaching the intracranial circulation¹⁾. The risks of intracranial stenting for atherosclerotic disease

have decreased with growing experience in the field, improved materials. The complications of the intracranial stenting include acute thrombosis, distal thromboembolism, arterial spasm, dissection with hemodynamic impact, guidewire perforation, and arterial rupture of the dilatation site⁵⁾. Some of these complications, such as vasospasm and dissection, are temporary and may result in minor neurological deficits. However, other complications, such as arterial rupture and acute thrombosis, are more serious and may result in neurologic sequelae or otherwise threaten the patient's life¹⁹⁾.

Certain factors associated with arterial dissection or rupture during percutaneous transluminal angioplasty or stenting are containing balloon size and its inflation pressure and velocity^{1,2,12,15)}. As our first case shown, the abrupt dilation of balloon and inappropriate inflation pressure has increased risk of vessel rupture. Balloon overdilation is not an accepted practice in the intracranial circulation, and it may be especially dangerous in the basilar artery and middle cerebral artery, where different authors advocate underdilation (no more than half of the normal diameter) because of the insubstantial muscularis and adventitial layers of the arterial wall and the subsequent elevated risk of perforation. In addition, constrained balloons have markedly higher internal pressure, which may lead to vessel rupture if balloons are much larger than the vessel diameter¹⁶⁾. Therefore, it is desirable that smaller size of balloon than diameter of vessel should be gradually dilated with lower pressure than nominal pressure^{1,13)}.

No firm data exist concerning the risk of the intracranial stenting as related to lesion location. In several series, there was no clear statistical result of which lesions are safer or of which are more dangerous. We found an 8.3% per-patient intracranial arterial complication rate during percutaneous revascularization procedures performed over a 3-year period, including a 3.3% rupture rate, an unpublished data. It appears that the primary risk is operator-dependent, followed closely by the risks related to location and type of lesion⁵⁾. Typical intracranial atherosclerotic disease presents in primarily five locations: the ICA carotid siphon, the main trunk of the middle cerebral artery, the distal vertebral artery, the vertebrobasilar junction, and the midportion of the basilar artery⁵⁾. The middle cerebral artery has a highest risk of the rupture or dissection in our series. In case 1, the patient died as a result of massive subarachnoid hemorrhage, although emergent balloon occlusion was achieved. We learned several valuable lessons from these complications we observed. Traditionally, the definitive treatment for traumatic intracranial artery rupture or pseudoaneurysm has been surgical repair⁴⁾. This treatment itself, however, is associated with an

unacceptable incidence of major complications, such as death and major stroke, and it is often an ineffective or even harmful treatment. In experience in peripheral circulation, the immediate treatment for acute iatrogenic rupture is balloon tamponade¹¹⁾. If failed of the initial balloon tamponade, placement of an additional stent may be effective because stent placement has been used effectively in the past to treat iatrogenic or traumatic rupture of the peripheral arteries^{6,14)}. Another patient was treated with still another ancillary technique, the placement of a stent-graft¹⁰⁾, after the initial tamponade procedure was not successful. Conceivably, a commercially manufactured stent-graft would be more readily available and easier to deploy³⁾. In conclusion, as our case has shown, patency can be quickly reestablished in acute rupture with additional stenting and balloon tamponade, even in the occurrence of thrombus.

Acute thrombosis is a potential complication of arterial angioplasty and stent placement. Because acute thrombosis is known to be platelet-rich thrombus, we elected to administer a platelet glycoprotein b/ γ antagonist as a rescue attempt to dissolve the clot. The potential mechanisms of this thrombolytic effect include active platelet deaggregation at the site of thrombus, endogenous autothrombolysis, and inhibition of platelet-rich thrombus propagation¹⁸⁾. Reports of the use of abciximab for neurovascular applications include its use as an adjunct to vertebrobasilar and carotid angioplasty, prevention of basilar artery rethrombosis after PTA, and rescue in acute carotid stent thrombosis¹⁷⁾. To our knowledge, this is the first report documenting abciximab use during an intracranial stenting procedure as successful rescue of thrombotic occlusion of the middle cerebral artery.

Conclusion

We describe two cases in which stenting of the MCA were complicated by acute rupture and thrombosis. One case was successfully controlled by endovascular treatment 3/4 an endovascular rescue not previously mentioned in the literature. Smaller size of balloon than diameter of vessel should be gradually dilated with lower pressure than nominal pressure for the prevention of intraprocedural rupture. If ruptured, urgent diagnosis and treatment of an iatrogenic injury of the intracranial artery is essential, as any delay may result in the patient's death. If acute extravasation of contrast material is encountered, balloon tamponade with an additional stenting is the immediate step that can be taken. Additionally, the intraarterial use of abciximab may be feasible and safe and it offers successful recanalization of acute thrombotic occlusions during the stenting.

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